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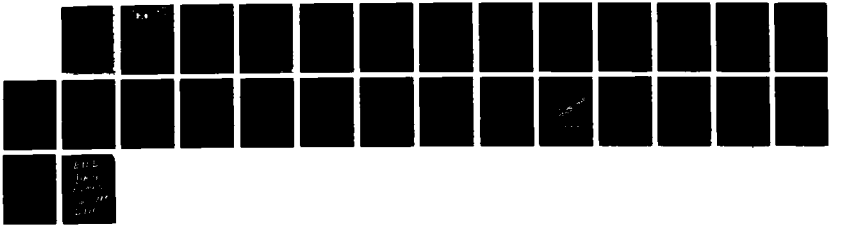

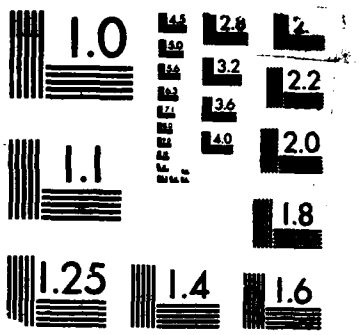

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This article reviews the effects of hot environments—and thus hyperthermia and hypohydration—on circulation, strength, endurance, and health in athletes. The cardiovascular responses to heat exposure at rest, and during exercise, are reviewed. Performance is reviewed by examining strength, power and indurance; the impact of hyperthermia, hypohydration and diuretic use on performance are discussed. The physiological needs for water, salt and carbohydrates have been examined. The four major heat illnesses (i.e. heat cramps, heat syncope, heat exhaustion, heatstroke) are described, as well as preventive measures to counteract hyperthermia and hypohydration. Keywords:

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THE IMPACT OF HYPERTHERMIA AND HYPOHYDRATION ON CIRCULATION, STRENGTH, ENDURANCE AND HEALTH.

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It should come as no surprise that heat exhaustion and heat stroke occur most often in the southern United States, during the months of May through September. Yet, many people do not realize that heat injuries also may occur in mild environments, when athletes wear uniforms, exercise at high intensities, and/or train in poorly ventilated areas. The purpose of this article is not to focus on environmental factors (i.e. air temperature, humidity, wind speed, clothing, solar energy), it is rather to focus on the physiological strain which results from exercise-heat exposure--especially in the forms of hyperthermia (elevated body temperature) and hypohydration (reduced body water).

At rest, the rectal temperature of the human body is maintained within a few tenths of a degree of 37°C (98.6°F). As the environmental temperature drops below 37°C, the blood flow to skin gradually decreases (to reduce heat loss) and shivering increases (to produce metabolic heat). As the environmental temperature rises above 37°C, skin blood flow increases (to dissipate stored heat) and sweating begins (to increase evaporative cooling).

Exercise assists the body when it is hypothermic (below 37°C) by producing heat, but is detrimental during hyperthermia (above 37°C), because (a) additional metabolic heat is produced and (b) exercising muscles compete with skin for the blood pumped by the heart (cardiac output). In fact, the cardiovascular system is fundamentally involved in man's ability to tolerate strenuous exercise in the heat and in the maintenance of body temperature. Impaired cardiovascular funtion becomes apparent during exercise in the heat, when sweat losses exceed 2% of body weight (17). If the cardiovascular system is overwhelmed, severe hyperthermia and heat injury occur (18).

Cardiovascular Responses to Heat

When resting humans are exposed to direct whole-body heating such as saunas (70 - 100°C, 158 - 212°F), their skin temperature rises. This is

immediately followed by a rise in skin blood flow, heart rate, and cardiac output. Because skin blood vessels dilate greatly in hot environments (Figure 1), the blood flow to muscles and internal organs decreases. This phenomenon also occurs prior to exercise in a hot environment.

FIG. 1

As exercise begins, the metabolic requirements of exercise superimpose further demands on the cardiovascular system. The pumping capacity (maximal cardiac output) of the average adult heart, during maximal exercise, is approximately 25 liters per minute (L/min); a liter is slightly larger in volume than 1 U. S. quart. In comparison, elite athletes may have maximal cardiac outputs of approximately 40 L/min. The ~~capacity~~ conductance of blood vessels in the skin and internal organs only totals approximately 10 L/min. However, because the ~~capacity~~ conductance of all muscle beds (60 - 70 L/min) easily exceeds the maximal cardiac output of the heart, cardiac output is a limiting factor during exercise (27).

FIG. 2

Figure 2 illustrates the impact which exercise has on blood flow in an average male (43.3°C, 110°F environment). The skin blood flow observed at rest (Fig. 1) decreases approximately 75% as exercise intensity reaches VO_2max (maximal oxygen consumption). This results in an increased central heat storage (hyperthermia), because blood does not flow to skin vessels adequately (27). Blood flow to internal organs changes very little as exercise intensity increases, leaving the greatest portion of cardiac output (80%) for exercising muscles--the site of the greatest metabolic demand. At VO_2max, total muscle blood flow increases approximately 800% (when compared to resting muscle blood flow).

FIG. 3

Figure 3 depicts the distribution of cardiac output at VO_2max, for untrained average males, trained average males, and for highly trained elite endurance athletes. The reader will notice that: (a) athletes may pump 60% more blood (17 L/min) than average males, (b) that training increases the

cardiac output of untrained males by approximately 10% (2 L/min), and (c) that the blood flow to skin and other organs is essentially the same in all three groups of males. Therefore, essentially all of the additional pumping capacity of an elite athlete (determined primarily by heredity) is presented to exercising muscles. This explains, in part, why athletes with high VO_2max values tend to be better able to tolerate exercise at high environmental temperatures (9).

Hyperthermia and Performance

Several studies have been conducted specifically to measure the effects of hyperthermia on muscle performance and exercise metabolism. In a review of animal research, Bennett (11) noted that the maximal force exerted by muscle tissue is relatively independent of muscle temperature. However, the most significant factor in movement is the rate of force application (power) and its removal. Maximal power output and relaxation speed are greatly influenced by muscle temperature, in all animal species which have been examined. Power output in animals is best represented by a bell-shaped curve, with maximal power occurring at muscle temperatures of 25 - 30°C (77 - 86°F). Muscle temperatures below and above this range (including hyperthermia) result in reduced maximal power output (29). Hyperthermia directly affects skeletal muscle by accelerating metabloic rate (known as the Q_{10} effect) to a point of inefficiency. The number of contractions to fatigue decreases, as muscle becomes hyperthermic (29).

In a study of sustained isometric muscle contractions, 28 - 32°C (82.4 - 89.6°F) was identified as the optimum human muscle temperature for endurance exercise of individual muscle groups. As muscle temperature increased above 32°C, endurance decreased; this was attributed to adverse metabolic changes, such as inadequate ATP levels and increased muscle cell acidity (14). Using a

different experimental design, Sargeant (28) immersed the legs of healthy men in a 44°C (111.2°F) water bath for 45 minutes, to measure changes in peak force and peak power during exercise on an isokinetic cycle ergometer. This resulted in an 11% increase of maximal peak force and power, when compared to resting trials at a room temperature of 21°C, 69.8°F. Immersing legs in 18°C (64.4°F) and 12°C (53.6°F) water baths resulted in peak force reductions of 12% and 21%, respectively. In addition, the magnitude of this temperature effect was dependent on pedalling rate.

The effects of temperature on endurance are different from the effects on strength and power. The optimal whole-body temperature for endurance, during aerobic dynamic exercise with large muscle groups, has not been precisely identified. However, lowering body temperature before exercise improves human endurance and prolongs exercise tolerance time markedly (21), by reducing cardiovascular strain and lowering blood lactic acid concentration. It has also been demonstrated that muscle glycogen depletion occurs at a lower rate at low rectal temperatures (15).

Hypohydration, Diuretics, and Performance

In this article, the term hypohydration is used to describe acute water loss, incurred over a few hours. The term dehydration is used to describe long-term water deficits of 24 hours or more.

A majority of studies have indicated that isometric strength and reaction time are not altered by hypohydration to -5% of body weight, although exceptions to these strength and reaction time studies have been reported (17). For example, Klinzing et al. (19) recently demonstrated that rapid hypohydration resulted in reduced scores on a wrestling performance test. The discrepancies among studies may be due to the method of hypohydration used.

ABLE 1 Table 1 describes the ratio of % change in plasma volume (%ΔPV) to %

change in body weight (%ΔBW), for several body weight loss techniques. A high ratio indicates that the %ΔPV is greater, proportionally, than the %ΔBW; it also predicts that cardiovascular strain will be high, and that cardiovascular function will be greatly affected. Of the body weight loss techniques shown in Table 1, diuretics place an individual at the greatest theoretical risk of heat exhaustion, because heat exhaustion is primarily a fluid volume depletion illness (see below). Also, if plasma volume losses are dependent on the weight loss technique, then it is likely that other body water compartments (e.g. intramuscular water) also vary from one technique to another.

At least two investigations have utilized diuretics to observe the effects of hypohydration on endurance performance. The first study (24) observed the effects of whole-body hyperthermia on cycling exercise tolerance in the laboratory. The authors concluded that pre-exercise passive heating, diuretic use, and prior exercise interact to reduce physical exercise capacity. The second study measured the performance of 8 healthy men (22 - 27 yr old), during 1500m, 5000m, and 10,000m foot races in mild environments (4). Blood plasma changes (after 5 hours of diuresis) averaged -9.9%, -12.3%, and -9.9%, respectively, while body weights changed -1.5 kg, -1.2 kg, and -1.6 kg, due to large urine losses. Figure 4 illustrates the mean running velocities of the normally hydrated (N) and hypohydrated (H) trials. The shortest race distance (1500m) was run 3.1% slower, after hypohydration. The 5000m and 10,000m distances were run 6.7% and 6.3% slower, respectively. Thus, the two highly aerobic endurance events appeared to be more effected by hypohydration than the event which involved a larger anaerobic component. This may be due to the fact that diuretics cause a greater reduction of blood plasma volume than the other methods of hypohydration shown in Table 1.

FIG. 4

The information above indicates that rapid weight loss via diuretics may be detrimental to bodily function and individual performance. Despite these facts, many wrestlers lose up to 9 kg (20 lb) in a brief period of time, in order to make a lower weight class. Weight loss is often accomplished by using laxatives or diuretic drugs. The American College of Sports Medicine (ACSM) has published a position stand regarding weight loss in wrestlers (3). This publication offers guidelines for safe body weight reductions, is available from ACSM, and should be in the library of every high school and college wrestling coach.

Water, Carbohydrate, and Salt Intake

Most of the aforementioned strength and endurance studies have examined the effects of hypohydration on performance in mild environments. When an athlete trains or competes in a hot environment, the effects of hypohydration on performance are magnified--especially at temperatures above 30°C (86°F). The beneficial effects of ingesting fluids during endurance activities in the heat have been unequivocally demonstrated (17).

Plain water is still the beverage of choice during most athletic contests. In events which last longer than 1 - 2 hours, however, dilute sugar additives (6 - 8% carbohydrate in solution) may be required to postpone the development of fatigue, to reduce muscle glycogen utilization, and to improve fluid-electrolyte absorption at the small intestine (23). During endurance exercise ranging from 22 - 51% of VO_2max, the utilization of ingested glucose is linearly related to relative exercise intensity (20); the higher the exercise intensity, the higher the glucose utilization from oral intake. The volume of fluid consumed by an athlete exerts the initial influence on stomach emptying rate, the caloric content exerts a secondary influence, and other factors (e.g. fluid temperature, sodium content, fat content, electrolyte concentration, anxiety, environmental conditions) interact thereafter to alter the gastric emptying rate (17,23).

The salt (sodium chloride, NaCl) requirements, of the vast majority of athletes training in the heat, can be met by liberally salting food during meals. The diet of the average U.S. citizen provides electrolytes in quantities which easily exceed the losses of electrolytes in sweat, urine, and feces (5). NaCl, potassium, or magnesium supplements are recommended only in situations where athletes lose copious quantities of sweat (6) each day (1 liter of water weighs 2.21 lb). For these reasons, salt tablets are not needed in the locker room or on the training field. In addition, an estimated 10 - 20% of all athletes experience stomach upset, nausea, or vomiting after consuming salt tablets.

The author knows of no evidence to indicate that salt supplements improve performance. Like vitamins, salt is necessary when a deficiency exists; but increased dietary salt does not necessarily improve performance. Although salt depletion heat exhaustion is a recognized heat illness (see below), the incidence of this form of heat exhaustion is very low among interscholastic athletes in North America. In fact, excessive salt intake (30g NaCl/day) may retard the normal course of heat acclimatization and may reduce maximal exercise capacity (13).

Heat Acclimatization (HA)

In considering the responses which the human body makes to consecutive days of exercise in a hot environment, it must first be recognized that the body adapts to heat exposure quite dramatically. However, there is little evidence to support the notion that the human body can adapt significantly to prolonged water deprivation. In other words, the body can adapt to heat, but not to dehydration.

Comparison of pre-HA to post-HA measurements shows that the following adaptations occur, when thermal stress and exercise intensity are constant: decreased heart rate, decreased rectal temperature, increased sweat rate,

decreased sweat NaCl loss, and expanded plasma volume (16). Although this HA response is technically termed "habituation", it will be termed "adaptation" here, for the sake of clarity. All of these adaptations improve heat tolerance because (a) heat transfer from the core to the periphery is improved and (b) because muscular exercise efficiency is improved (this is not gained by physical training alone).

Most HA adaptations are established during the first week of heat exposure, and not all of these adaptations occur at the same rate. During the initial 3 - 6 days of HA, adaptations occur primarily in the cardiovascular system; plasma volume expands and heart rate decreases. The consumption of large quantities of water during heat acclimatization will insure cardiovascular stability during this period. Rectal temperature requires 5 - 8 days to stabilize at a new lower level, while sweat NaCl concentration (6 - 10 days) and sweat rate (10 - 12 days) require longer to stabilize (5). The majority of HA adaptations will dissipate in 14 - 21 days, if not maintained.

To insure optimum heat acclimatization, rectal temperature must be elevated to a safe level (38 - 39°C, 100.6 - 102.2°F) and maintained for approximately 90 minutes each day, for 10 - 14 days. During prolonged exercise, intensity should be maintained at 50% of VO_2max or higher. Passive heat exposure may induce partial HA, but it cannot be expected to match the results gained by exercising in the heat (9,25). However, coaches and athletes are cautioned that any attempt to induce hyperthermia by wearing excessive clothing can be dangerous. Rubber sweat suits or insulated garments may result in excessive heat storage, heat exhaustion, or heatstroke. Large areas of skin surface should be left exposed to the air, to allow adequate heat dissipation by radiation, conduction, convection and evaporation.

With this precaution in mind, two successful cases of artificial HA will be described. First, distance runner Ron Daws prepared for the 1967 Holyoke (MA) Marathon (36°C, 97°F) by wearing a sweat suit during his spring and summer training runs. Although Daws was not expected to be among the leaders, he won this National A.A.U. Championship because his opponents were not heat acclimated (22). Second, the University of Nebraska football team prepared for its September, 1987 contest at Arizona State by utilizing a unique form of artificial HA. After considering the pros and cons of wearing sweat suits in the heat (see previous paragraph), a member of the Nebraska coaching staff contacted the U.S. Army Research Institute of Environmental Medicine, Natick, MA, for advice. The coaching staff then decided to create a "heat chamber" in Shulte Field House by increasing the air temperature to approximately 37°C (98.6°F). Players ran short sprints and rested between sprints, to insure that rectal temperatures were kept at safe levels. The Cornhuskers were also fortunate in that they had a week in their schedule without a contest; this allowed 12 consecutive days of workouts in the heat, whereas 5 - 7 days would have provided only partial HA. During the first few days many football players experienced headaches and found training to be more difficult than usual. By the 10th - 12th day, however, they found practices to be much like their usual training sessions.

The Medical Threat of Hyperthermia and Hypohydration

The headaches and fatigue which these football players experienced (above) are commonly seen in athletes during the first 1 - 5 days of heat exposure or during a heat wave. Thus, an understanding of the four major heat illnesses (symptoms and treatment) is vital for the coach and athlete alike (5,18). Table 2 summarizes the signs and symptoms characteristic of desert hypohydration and dehydration (1). Clearly, as body water decreases, these

TABLE 2

characteristics increase in severity. The signs and symptoms described in Table 2 may, or may not, be observed as a part of the four major heat illnesses (i.e. heat cramps, heat syncope, heat exhaustion, heatstroke).

The exact cause of heat cramps (or other maladies such as exercise-induced muscle cramps and nocturnal cramps) is unknown. They usually occur after several hours of strenuous exercise in individuals who have lost a large volume of sweat, who have drunk a large volume of unsalted water, and who have excreted a small volume of urine (31). Heat cramps usually occur in the muscles of the legs, arms, and abdomen, but have been confused with gastro-intestinal upset and hyperventilatory spasms of the hands and feet. Although the pain of heat cramps is excruciating, treatment is usually rapid, effective, and without complications. Treatment of heat cramps focuses on the fact that victims have reduced blood and urine levels of NaCl (18). A 0.1% oral NaCl solution (i.e. 1g or two 10 grain salt tablets per 1 L water) is recommended for mild heat cramps. Intravenous salt solutions (i.e. 0.5 - 1.0 L normal saline) are utilized in severe cases.

Heat syncope (fainting) occurs when there has been a sudden rise in environmental temperature or humidity, and is observed most often during the first 3 - 5 days of heat exposure (see the cardiovascular adaptations of heat acclimatization above). It occurs when a large volume of blood flows to dilated skin blood vessels, postural pooling of blood, diminished return of blood to the heart, and lack of blood flow to the brain. Treatment involves lying in a supine position in a shaded area, and avoidance of sudden or prolonged standing. Assessment of hypohydration and replacement of fluid deficits are also important. Heat syncope is usually categorized as a syndrome separate from heat exhaustion.

Heat exhaustion is the most common heat illness among athletes, and is usually divided into two categories: salt-depletion and water-depletion heat exhaustion. In reality, these forms of heat exhaustion rarely occur alone, because body fluid losses (sweat and urine) contain both salt and water. The symptoms of these two forms of heat exhaustion are numerous, but urine and blood NaCl levels seem to be the only consistent distinguishing criteria (18). Heat exhaustion includes various combinations of the following signs and symptoms: flushed skin on head and torso with "heat sensations", chills, abdominal cramps, goose flesh, elevated heart rate at rest, "rubbery" legs, vomiting, hyperirritability, headache, dizziness, fatigue, anxiety, hyperventilation, diarrhea, nausea, syncope, and heat cramps (7,8). Heat exhaustion is primarily a hypohydration or dehydration problem; rapid recovery occurs when intravenous fluids are administered. Normal, young heat exhaustion victims may require up to 4 liters of intravenous fluid after prolonged endurance exercise (12). Cooling the skin with water or ice will constrict blood vessels, thereby improving blood flow to the brain and other internal organs.

Heatstroke is the second leading cause of death among interscholastic athletes (head and spinal injuries is the leading cause). The frequency of death by heatstroke (mortality range: 10 - 80%) is related to the duration and intensity of whole-body hyperthermia (over 40°C, 104°F), which may damage every organ in the body. Heatstroke may or may not involve the symptoms of heat exhaustion; yet, an unconscious heat exhaustion victim can rapidly become a heatstroke victim, if left untreated. Table 3 describes characteristics which are commonly used to distinguish heat exhaustion from heatstroke. Sweating may or may not be present in heatstroke. Some heat illness patients have symptoms of both heat exhaustion and heatstroke; these patients should be treated as though they have heatstroke, and should be cooled immediately (12).

TABLE 3

Upright exercise is invariably involved with heatstroke in athletes. Our research on former heatstroke patients indicates that exercising individuals are relatively unaware of the seriousness of their hyperthermia, in comparison to the familiar sensations of fatigue or exhaustion (7). Very few cases of heatstroke have been reported among cyclists (i.e. rapid air movement across exposed skin), in spite of high exercise intensities and long durations. Treatment of hyperthermia consists of cooling the body via cool water and/or ice, and fanning. Cooling must continue during evacuation to the nearest hospital, where treatment can be continued by a physician.

Counteracting the Environmental Heat Threat

By employing the following guidelines, the coach/practitioner can optimize performance and well-being:

1. Know the signs and symptoms of the four major heat illnesses. Be prepared to treat heat injury. Organize first-aid supplies and body cooling techniques in advance.
2. Cool water sprays or showers reduce rectal temperature only a few tenths of a degree, if exercise continues (10). Spraying or showering is only beneficial for the individual who discontinues exercise (e.g. halts internal heat production).
3. Move water supplies near to exercising individuals. Provide water which is chilled, plentiful and palatable.
4. Supervise daily body weight measurements rigidly. Remove athletes who report for training with 2% (or more) body weight loss (30). This should include tournaments which involve successive days of competition (i.e. track and field, wrestling, tennis, soccer, basketball, swimming).
5. Follow published guidelines (3) for weight loss by wrestlers. Discourage diuretic and/or laxative use.

6. Instruct athletes to monitor their own urine volume and color. Ideally, urine output should total 1.5 liters per day (or more). Urine color should be pale yellow, except shortly after exercise.
7. Expose athletes to heat gradually, for 10 - 14 days. The first 5 days of heat acclimatization are critical for cardiovascular stability. Provide specific written instructions regarding heat acclimatization, prior to initial workouts or attending a summer camp.
8. Instruct athletes to salt food liberally during meals, especially during the first 5 days of heat acclimatization. Discourage the use of salt tablets.
9. Discontinue training and competition whenever temperature and humidity warrant (2). Select clothing and design training (e.g. intensity, frequency, mode, duration) each day using temperature and humidity data.
10. Recognize that the following individuals are at increased risk of heat injury: preadolescents, poorly conditioned, unacclimatized, those taking medications (e.g. blood presure or cold/hay fever medication), those with skin disorders which limit sweating, those with high body weight per unit of surface area (5).

References

1. Adolph, E.F. (1947) Physiology of Man In the Desert. Interscience Pub Inc. New York, NY.
2. American College of Sports Medicine. (1987) Position stand on: Prevention of thermal injuries during distance running. Med Sci Sports Exerc, 19, 529-533.
3. American College of Sports Medicine. (1976) Position stand on: Weight loss in wrestlers. Med Sci Sports, 8, XI-XII.
4. Armstrong, L.E., Costill, D.L., and Fink, W.J. (1985) Influence of diuretic-induced dehydration on competitive running performance. Med Sci Sports Exerc, 17, 456-461.
5. Armstrong, L.E. and Dziados, J.E. (1986) Effects of heat exposure on the exercising adult, pp. 17-216. In: Sports Physical Therapy, Bernhardt D. (Ed). Churchill Livingstone Pub Co. New York, NY.
6. Armstrong, L.E., Hubbard, R.W., Jones, B.H. and Daniels, J.T. (1986) Preparing Alberto Salazar for the heat of the 1984 Olympic marathon. Physician Sportsmed, 3, 73-81.
7. Armstrong, L.E., Hubbard, R.W., Kraemer, W.J., DeLuca, J.P. and Christensen, E.L. (1988) Signs and symptoms of heat exhaustion during strenuous exercise. Ann Sports Med, in press.
8. Armstrong, L.E., Hubbard, R.W., Szlyk, P.C., Sils, I.V., and Kraemer, W.J. (1988) Heat intolerance, heat exhaustion monitored: a case report. Aviat Space Environ Med, in press.
9. Armstrong, L.E. and Pandolf, K.B. (1988) Physical training, cardiorespiratory physical fitness and exercise-heat tolerance, chapter 5. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes, Pandolf K.B. (Ed) Benchmark Press. Indianapolis, IN.

10. Bassett, D.R., Nagle, F.J., Mokerjee, S., Darr, K.C., Ng, A.V., Voss, S.G. and Napp, J.P. (1987) Thermoregulatory responses to skin wetting during prolonged treadmill running. Med Sci Sports Exerc, 19, 28-32.

11. Bennet, A.F. (1984) Thermal dependence of muscle function. Am J Physiol, 247, R217-R229.

12. Callaham, M.L. (1979) Emergency Management of Heat Illness. Abbott Laboratories. North Chicago, IL.

13. Dassler, A.R., Karas, S., Bowman, J.S. and Hardenbergh, E. (1973) Adverse effects of supplementary sodium chloride on heat adaptation. Fed Proc, 32, 336A.

14. Edwards, R.H.T., Harris, R.C., Hultman, E., Kayser, L., Koh, D. and Nordesjo, L.O. (1972) Effect of temperature on muscle energy metabolism and endurance during successive isometric contractions. J Physiol (London), 220, 335-353.

15. Fink, W.J., Costill, D.L. and VanHandel, P.J. (1975) Leg muscle metabolism during exercise in the heat and cold. Eur J Appl Physiol, 34, 183-190.

16. Greenleaf, J.E. and Greenleaf, C.J. (1970) Human acclimation and acclimatization to heat: a compendium of research. NASA Technical Report No. TM X-62008. Ames Research Center, Moffett Field, CA.

17. Herbert, W.G. (1983) Water and electrolytes, pp 56-98. In: Ergogenic Aids in Sport, Williams M. (Ed). Human Kinetics Pub Co. Champaign, IL.

18. Hubbard, R.W. and Armstrong, L.E. (1988) The heat illnesses: biochemical, ultrastructural, and fluid-electrolyte considerations, chapter 8. In: Human Performance Physiology and Environmental Medicine at Terrestrial Extremes, Pandolf K.B. (Ed) Benchmark Press. Indianapolis, IN.

19. Klinzing, J.E. and Karpowicz, W. (1986) The effects of rapid weight loss and rehydration on a wrestling performance test. J Sports Med, 26, 149-156.

20. Krzentowski, G., Jandrsam, B.V., Pirnay, F., Mosora, F., Lacroix, M. Luyckx, A.S., and Lefebvre, P.J. (1984) Availability of glucose given orally during exercise. J Appl Physiol, 56, 315-320.

21. MacDougall, J.D., Reddan, W.G., Layton, C.R. and Dempsey, J.A. (1974) Effects of metabolic hyperthermia on performance during heavy prolonged exercise. J Appl Physiol, 36, 538-544.

22. Mirkin, G. and Hoffman, M. (1978) The Sportsmedicine Book. Little, Brown and Co. Boston.

23. Murray, R. (1987) The effects of consuming carbohydrate-electrolyte beverages on gastric emptying and fluid absorption during and following exercise. Sports Med, 4, 322-351.

24. Nielsen, B., Kubica, R., Bonnesen, A., Rasmussen, B., Stoklosa, J. and Wilk, B. (1981) Physical work capacity after dehydration and hyperthermia. Scand J Sports Sci, 3, 2-10.

25. Pandolf, K.B. (1979) Effects of physical training and cardiorespiratory physical fitness on exercise-heat tolerance: recent observations. Med Sci Sports, 11, 60-65.

26. Rowell, L.B. (1974) Human cardiovascular adjustments to exercise and thermal stress. Physiol Rev, 54, 75-159.

27. Rowell, L.B. (1986) Human Circulation Regulation During Physical Stress. Oxford University Press. New York, NY.

28. Sargeant, A.J. (1987) Effect of muscle temperature on leg extension force and short-term power output in humans. Eur J Appl Physiol, 56, 693-698.

29. Segal, S.S., Faulkner, J.A., White, T.P. (1986) Skeletal muscle fatigue in vitro is temperature dependent. J Appl Physiol, 61, 660-665.

30. Smith, N.J. (1984) The prevention of heat disorders in sports. Am J Diseases Children, 138, 786-790.

31. Talbot, J.H. (1935) Heat Cramps. In: Medicine. Williams and Wilkins Co. Baltimore.

32. Veghte, J.H. and Webb, P. (1961) Body Cooling and Response to Heat. J Appl Physiol, 16: 235-238.

Figure Legends

Figure 1 — Distribution of cardiac output (L/min) at rest, in cool and hot environments. Redrawn from Rowell (27).

Figure 2 — Distribution of cardiac output in a 43.3°C (110°F) environment, at exercise intensities ranging from rest to VO_2max. Redrawn from Rowell (27).

Figure 3 — Distribution of cardiac output in three groups of males, during brief, maximal exercise. Redrawn from Rowell (27).

Figure 4 — Running Velocities of eight males, at 1500 m, 5000 m, and 10000 m. Trials were run in mild environmental conditions, while normally hydrated (N) and hypohydrated (H). Redrawn from Armstrong et al. (4).

FIGURE 1

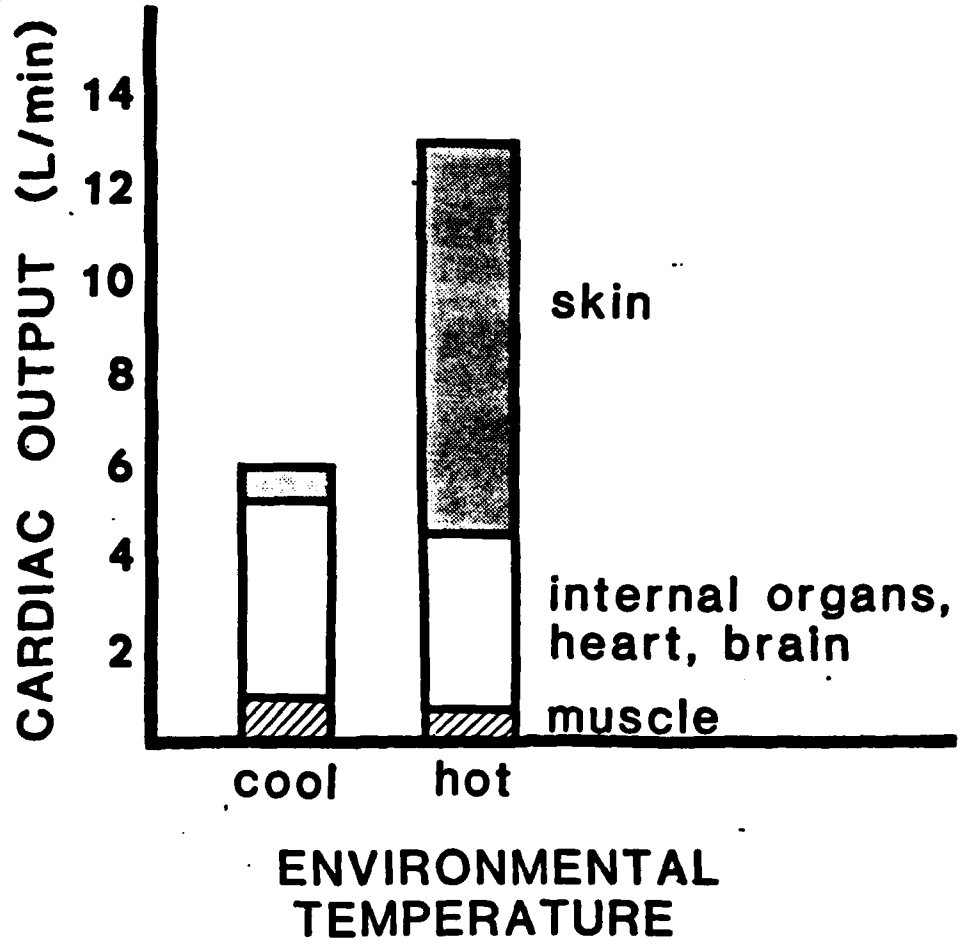
CARDIAC OUTPUT (L/min)
14
12
10
8
6
4
2
skin
internal organs,
heart, brain
muscle
cool
hot
ENVIRONMENTAL
TEMPERATURE

FIGURE 2

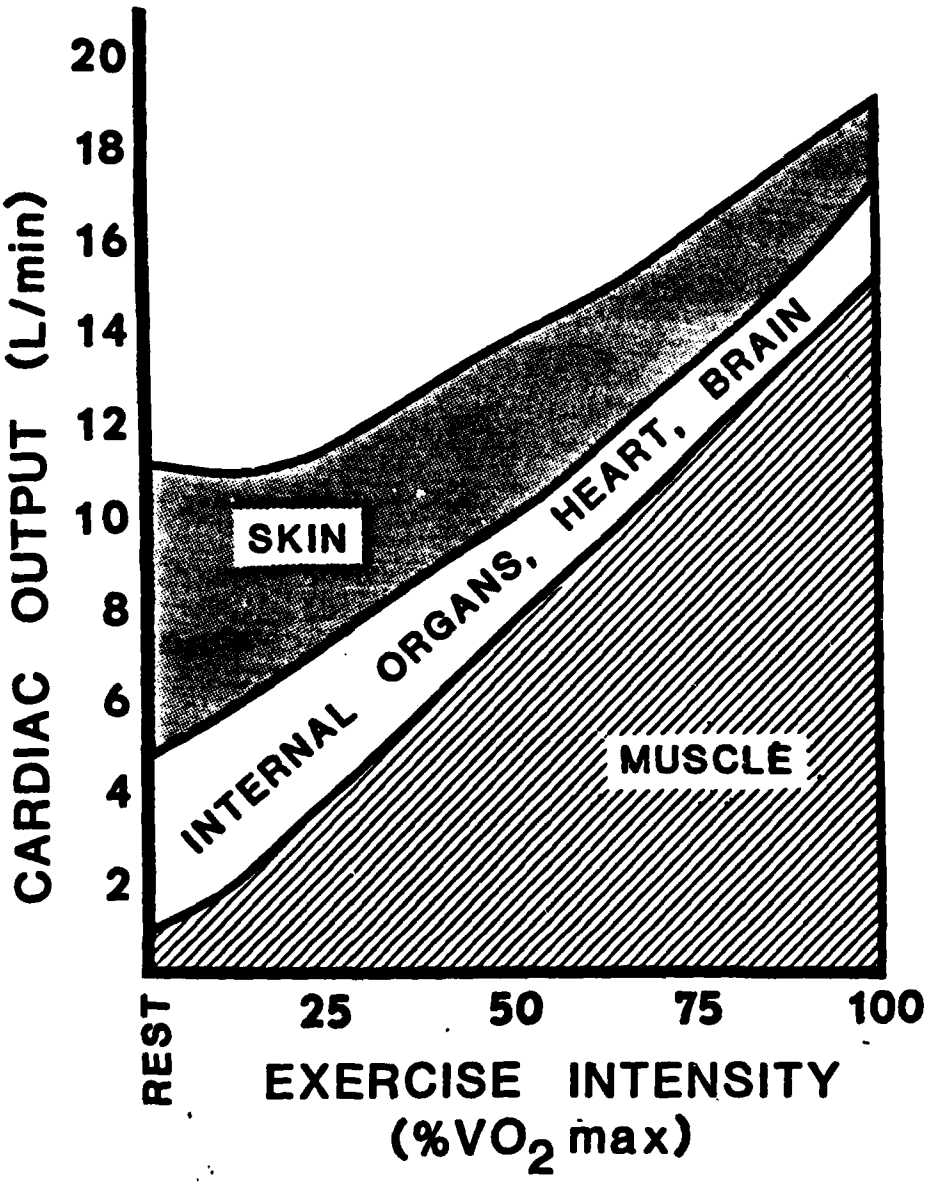
CARDIAC OUTPUT (L/min)
20
18
16
14
12
10
8
6
4
2
SKIN
INTERNAL ORGANS, HEART, BRAIN
MUSCLE
REST
25
50
75
100
EXERCISE INTENSITY
(%VO2 max)

FIGURE 3

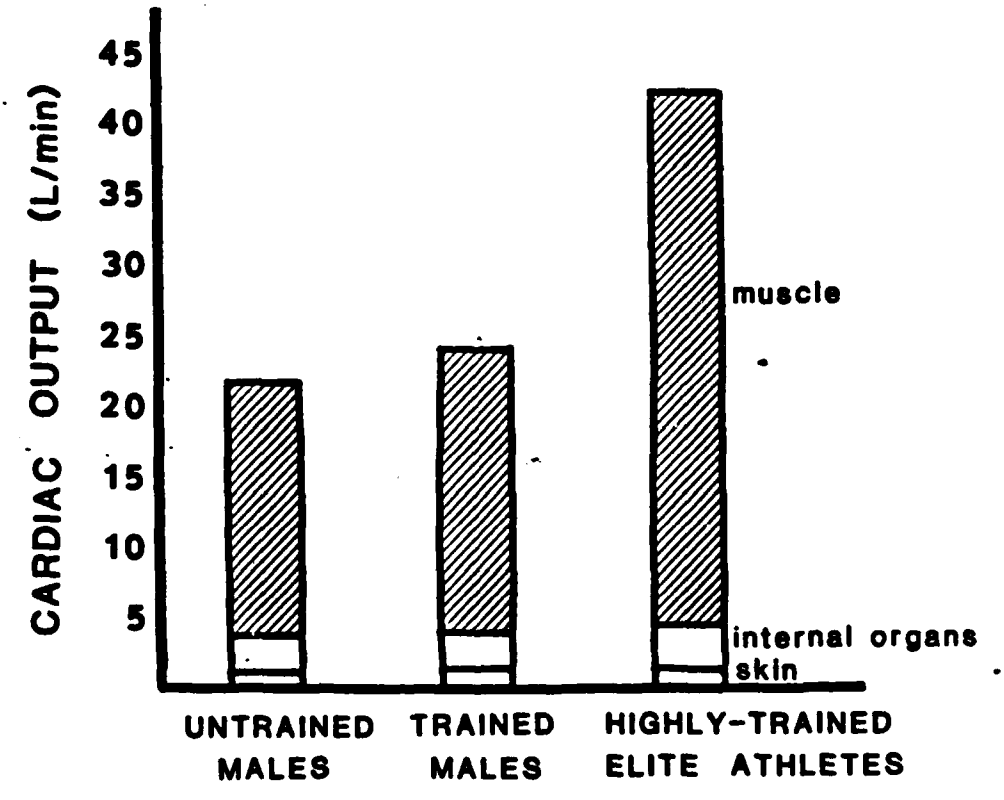
CARDIAC OUTPUT (L/min)
45
40
35
30
25
20
15
10
5
muscle
internal organs
skin
UNTRAINED MALES
TRAINED MALES
HIGHLY-TRAINED ELITE ATHLETES

FIGURE 4

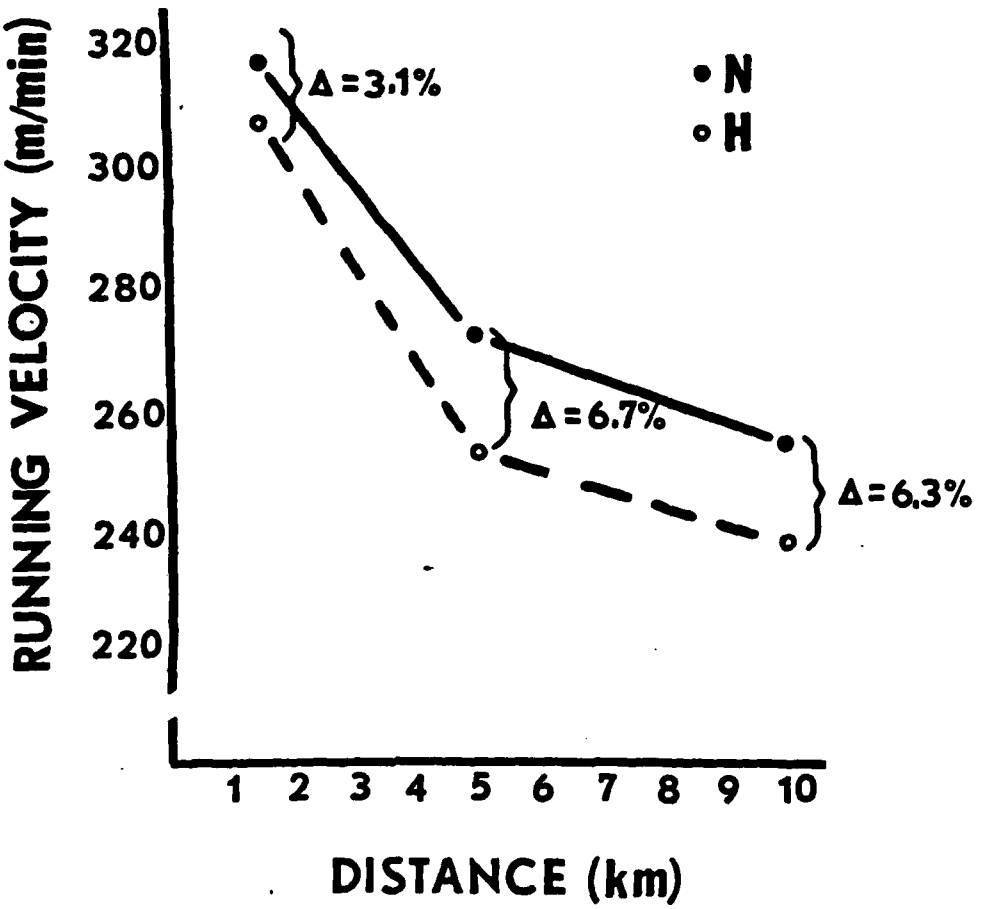
RUNNING VELOCITY (m/min)
320
300
280
260
240
220
Δ=3.1%
Δ=6.7%
Δ=6.3%
• N
○ H
1 2 3 4 5 6 7 8 9 10
DISTANCE (km)

Table 1 - Effects of weight loss technique on the percentage plasma volume change (%ΔPV)-to- percentage body weight change (%ΔBW) ratio (18). A high ratio indicates that the percentage of plasma volume loss is greater than the percentage of body weight loss.

Technique	Ratio of %ΔPV/%ΔBW
diuretic use	5:1 to 6:1
passive thermal hypohydration	4:1 to 5:1
exercise in a cool environment	3:1 to 4:1
exercise in a hot environment	2:1 to 3:1
witholding fluids and food for 48 hours	1:1

Table 2 - Signs and symptoms of hypohydration and dehydration in the desert. Information originally reported by Adolph (1).

Body water loss (% of initial weight)	Signs and symptoms
0-2	none
2-4	thirst, vague discomfort, loss of appetite, verbal complaints, reduced movement
4-6	slowing of march pace, oppressed by heat, flushed skin, tingling sensation in arms/neck/back, apathy, impatience, weariness, sleepiness
6-8	cotton-mouth, stumbling, headache, dizziness, indistinct speech, blue coloration in skin and lips, shortness of breath
8-12	spasticity, delerium, wakefulness, swollen tongue
12-15	inability to swallow, shriveled tongue, sunken eyes, dim vision, painful urination
15-20	stiffened eyelids, deafness, numb skin, cracked skin, inability to urinate, death

Table 3 - Factors which distinguish heat exhaustion from heatstroke (18).

FACTOR	HEAT EXHAUSTION	HEATSTROKE
rectal temperature	below 104°F	above 104°F
blood enzymes*	normal	elevated
mental status	normal	disturbed
spontaneous cooling	present	usually absent

* - CPK, ALT, AST, or LDH

END

DATE

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